CASE

A 73-year-old male with hypertension, chronic kidney disease, coronary artery disease, and Parkinson's disease presented to the emergency department with fever and diarrhea. His temperature was 38.4°C, heart rate 106 bpm, and blood pressure 138/75 mmHg, with atrial fibrillation and signs of heart failure. The Burch-Wartofsky score was 50, consistent with thyroid storm.

Laboratory tests revealed free thyroxine 4 37.8 pmol/L (NR 11.5–22.7), free thyroxine 3 5.6 pmol/L (NR 3.5–6.5), and thyroid-stimulating hormone <0.01 mIU/L (NR 0.55–4.78). Thyroid autoantibodies, including anti-thyroid peroxidase, anti-thyroglobulin, and thyroid-stimulating immunoglobulins, were negative (<0.10 IU/L). The thyroid storm was precipitated by invasive *Klebsiella* syndrome with endophthalmitis and lung and liver abscess. He was treated with Lugol's iodine, corticosteroid, antibiotics, and carbimazole.

Ultrasound thyroid revealed a mixed cystic-solid nodule in the left thyroid lobe, measuring 2.3 × 3.3 × 4.3 cm (TI-RADS category 3). Technetium-99m thyroid scintigraphy demonstrated a hyperfunctioning left thyroid nodule with a focal intranodular cold spot measuring 5.0 × 3.7 cm. Fine needle aspiration cytology of the nodule was benign follicular cells. Following stabilization with anti-thyroid treatment, he underwent left hemithyroidectomy. Histopathology examination revealed a Hurthle cell adenoma without capsular or vascular invasion.

Postoperatively, he remained clinically euthyroid. Surveillance ultrasound performed 8 months later showed a normal right thyroid lobe, and lifelong surveillance was planned.

CONCLUSION

This case illustrates a rare and unusual presentation of thyroid storm caused by a toxic Hurthle cell adenoma containing an intranodular cold spot on scintigraphy. To our knowledge, only one similar case has been reported in the literature, and our case is the first to present with thyroid storm.

EP_A152

ACUTE THYROID PAIN IN PREGNANCY: PAINFUL HASHIMOTO'S THYROIDITIS VERSUS SUBACUTE THYROIDITIS— A CASE REPORT

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INTRODUCTION

Painful Hashimoto's thyroiditis is a rare and atypical form of autoimmune thyroid disease characterized by thyroid pain and tenderness. It can closely resemble subacute thyroiditis and must be distinguished from other causes of thyroid pain, including hemorrhage into a cyst and thyroid abscess. This distinction is particularly important in pregnancy, where radionuclide imaging is not recommended.

CASE

A 27-year-old pregnant female (G4P3) at 24 weeks' gestation, with Southeast Asian ovalocytosis and diet-controlled gestational diabetes, was diagnosed with primary hypothyroidism at 14 weeks following evaluation of a painless goiter. Initial tests showed markedly elevated thyroid-stimulating hormone (>150 mIU/L), low free thyroxine (3.5 pmol/L), and positive anti-thyroid peroxidase antibodies (319 IU/mL), consistent with Hashimoto's thyroiditis. Levothyroxine therapy achieved biochemical euthyroidism.

At 24 weeks, she developed acute right-sided anterior neck pain radiating to the ear, with dysphagia, odynophagia, and fever. Examination revealed a tender thyroid without lymphadenopathy. Ultrasound demonstrated diffuse enlargement with bilateral ill-defined hypoechoic avascular areas. Inflammatory markers showed markedly elevated C-reactive protein (184 mg/L) with a normal erythrocyte sedimentation rate (16 mm/h), while thyroid function remained within target range. Imaging and clinical findings made abscess and hemorrhage unlikely.

The presence of pre-existing autoimmune thyroid disease, antibody positivity, and hypothyroidism before symptom onset supported painful Hashimoto's thyroiditis over subacute thyroiditis. This case highlights that normal thyroid function tests do not necessarily indicate disease remission, as inflammatory activity may persist independently of hormone levels.

CONCLUSION
Painful Hashimoto's thyroiditis should be considered in pregnant patients with known autoimmune thyroid disease presenting with acute thyroid pain despite normal thyroid function. Diagnosis requires integration of clinical, biochemical, and imaging findings to guide appropriate management.

EP_A153

THE HIDDEN HAZARD AFTER TOTAL PARATHYROIDECTOMY: A CASE REPORT

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INTRODUCTION
Palpation thyroiditis is an underrecognized condition that occurs after parathyroidectomy. Symptoms range from mild to overt thyrotoxicosis that may result in life-threatening cardiac arrhythmias and acute coronary syndrome. Diagnosis is made by having suppressed thyroid-stimulating hormone (TSH) and elevated free thyroxine 4 (fT4) or free thyroxine 3 (fT3) and low radionuclide uptake on thyroid scan.

CASE
We report a case of a 72-year-old female with underlying end-stage renal disease on regular hemodialysis who underwent total parathyroidectomy for tertiary hyperparathyroidism. Her thyroid function test (TFT) preoperatively was normal, and cardiac assessment showed normal echocardiogram findings and sinus rhythm on electrocardiogram. Her operation was uncomplicated, involving neck exploration and all 4-gland removal. Postoperatively, she had new-onset atrial fibrillation (AF) with rapid ventricular response at day 5, requiring electrical and chemical cardioversion. She had recurrent tachyarrhythmias on day 6. Her repeated TFT showed suppressed TSH, 0.036 mIU/L (NR 0.38–5.33) and raised fT4, 35.4 pmol/L (NR 7.9–14.4), fT3–fT4 ratio 0.26 (<0.3), consistent with thyrotoxicosis. She was treated as thyroid storm (Burch-Wartofsky Point Scale 45), after ruling out other causes. She was started on glucocorticoids, propylthiouracil, and Lugol's iodine. Despite biochemical improvement, she continued to develop recurrent AF up till Day 12 post-op, requiring multimodal treatment, including beta-blocker and antiarrhythmic agents. She was later discharged well on low-dose carbimazole. Her ultrasound neck showed a normal thyroid gland, and her thyroid antibodies were negative.

CONCLUSION
A high level of suspicion is needed to detect thyroiditis post-parathyroidectomy due to its potential adverse impact on surgical outcomes. Treatment is mainly symptomatic, and the disease is self-limiting. Risk factors identified include secondary/tertiary hyperparathyroidism and bilateral neck exploration that involves manipulation of the thyroid gland.

EP_A154

SUBACUTE HYPOTHYROID MYOPATHY AS AN ATYPICAL PRESENTATION FOLLOWING RADIOIODINE THERAPY

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INTRODUCTION
Hypothyroidism is the most common outcome following radioiodine (RAI) therapy for Graves' disease, affecting up to 80% of patients, usually within 6 months. While symptoms are often nonspecific, musculoskeletal complaints may be the predominant or sole manifestation. Hypothyroid myopathy occurs in 30–80% of patients, typically causing myalgias, cramps, fatigue, and slowly progressive, symmetric proximal weakness with delayed reflex relaxation. We report an atypical case with subacute and evolving weakness after levothyroxine initiation.

CASE
A 43-year-old female with Graves' disease underwent RAI therapy (25 mCi) and developed hypothyroidism 7 weeks later. She was started on levothyroxine 50 mcg daily. Two weeks into treatment, she presented with progressive proximal lower limb weakness (power 4/5), while distal strength and reflexes remained intact. Labs revealed elevated creatine kinase (259 U/L), hypokalemia (3.3 mmol/L), creatinine (57 µmol/L), and severe hypothyroidism (thyroid-stimulating hormone [TSH] 52.88 mIU/L, free thyroxine 4 [FT4] 7.79 pmol/L). Levothyroxine was increased to 100 mcg daily. Two weeks later, she developed proximal upper limb weakness (power 4/5), while lower limb strength had normalized. Nerve conduction studies and electromyography were unremarkable. Labs showed creatine kinase (244 U/L) and creatinine (58 µmol/L). As she remained hypothyroid (TSH 20.85 mIU/L, FT4 11.61 pmol/L), levothyroxine 100 mcg daily was continued. Her symptoms gradually improved alongside biochemical recovery (TSH 8.83 mIU/L, FT4 15.90 pmol/L) after 4 weeks, consistent with hypothyroid myopathy.